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In recent years evidence has been appearing in the literature indicating that the immune response may be playing an important role in the initiation and progression of the periodontal diseases. It was felt that research should be continued in this area, and a study was initiated to determine the IgA, IgG, and IgM concentrations in the granulation tissue removed from deep infrabony pockets of patients with periodontosis and advanced periodontitis. The data revealed a considerable variability in concentrations of the immunoglobulins. Comparison

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II. CONCENTRATIONS OF IMMUNOGLOBULINS IN GRANULATION TISSUE FROM POCKETS OF PERIODONTOSIS AND PERIODONTITIS PATIENTS

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(10) RONALD L. VAN SWOL, D.D.S.*
ARTHUR GROSS, D.D.S., M.S.†
JEAN A. SETTERSTROM, B.S., M.S., Ph.D.§
SANDRA M. D'ALESSANDRO, B.S.§

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*Commander, Dental Activity
Walter Reed Army Medical Center
Washington, D. C.

†Chief, Division of Oral Biology
U. S. Army Institute of Dental Research
Walter Reed Army Medical Center
Washington, D. C.

§Division of Oral Biology
U. S. Army Institute of Dental Research
Walter Reed Army Medical Center
Washington, D. C.

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IMMUNOGLOBULINS IN PERIODONTAL TISSUES

II. CONCENTRATIONS OF IMMUNOGLOBULINS IN GRANULATION TISSUE FROM POCKETS OF PERIODONTOSIS AND PERIODONTITIS PATIENTS

Since 1923 when Gottlieb[1] first introduced the concept of a non-inflammatory degenerative disease of the supporting tissues of a tooth and 1942 when Orban and Weinmann[2] named this clinical condition "periodontosis," a great deal of effort has been expended in attempting to elucidate the etiology of this disease. Generally, systemic diseases and syndromes have been implicated as possible etiologic factors that influence and mediate the severe periodontal destruction seen in these cases. In all instances, however, no "cause and effect" relationship has ever been established.

For many years then, the assumption that periodontosis was a degenerative noninflammatory entity has been in vogue. It certainly can exist with little or no visual clinical sign of gingival inflammation.[3-5] However, many cases are seen by the dentist at a point in time where inflammation is a prominent finding.[6] Since it has been shown that inflamed gingiva, including gingiva from patients with periodontosis, contain a greater number of plasma and lymphoid cells than normal tissue (especially in the deep aspects of the pocket next to the bone)[7,8] it is becoming clear that the old definition of periodontosis, as a degenerative, rather than an inflammatory process, is in need of revision. We support the suggestion of Baer and Kaslick,[9] however, who have recommended that until sufficient knowledge is available to correctly name this disease it should be designated "Periodontosis (Juvenile Periodontitis)."

In the last decade data have emerged supporting the concept that periodontal diseases are the result of the interaction of oral

bacteria and the host's periodontal tissues. The immune response then, is now considered to play an important role in the initiation and progression of periodontal disease (see review[10]).

In regard to periodontosis, recent evidence indicates the presence of a clinico-pathologically distinct, rapidly progressive disease in the adolescent that is different from chronic adult periodontitis in that it manifests a deficient *in vitro* cell-mediated response to some gram negative bacteria involved in the pathogenesis of the disease.[11-12] Based on the evaluation of neutrophil chemotaxis in nine patients with juvenile periodontitis, Clark, Page, and Wilde[13] concluded that the tissue destruction seen was in part the result of a failure of the host defense mechanism. Findings by Budtz-Jörgenson *et al.*[14] tend to support the assumption that a state of immunodeficiency can be implicated in the etiology of periodontosis, as do the scanning electron micrographs of Freedman *et al.*[15] who examined the surface morphology of lymphocytes isolated from periodontosis lesions and found them to have smooth surfaces, indicating that they were not activated.

Although there have been numerous reports concerning the cellular immune response of individuals with periodontosis[11-13] and studies demonstrating defective neutrophil function and chemotaxis[13,16,17] there have been few findings published regarding the humoral response and local tissue immunoglobulin levels. Abundant immunoglobulin-containing cells have been observed in periodontosis tissues[7] and increased concentrations of IgA, IgG, and IgM have been reported in the serum of periodontosis patients.[11] The serum IgM concentrations were found to be

increased to a greater extent than those of IgG and IgA. Complement activation via the alternate pathway in the gingival pocket fluid of periodontosis patients has been demonstrated, whereas, no such reaction has been shown to occur in the serum.[18]

In the most recent study by Gross *et al.*[19] immunoglobulin concentrations in inflamed gingiva from patients with moderate periodontitis and in normal gingiva were determined. The results revealed an increase in IgA and IgG levels as the degree of inflammation increased. IgM was not consistently detectable.

Since no information concerning the quantitative immunoglobulin content in the tissue from periodontosis patients is available, it was the purpose of this study to determine the IgA, IgG, and IgM concentrations in the granulation tissue removed from deep infrabony pockets of patients with periodontosis and advanced periodontitis.

MATERIALS AND METHODS

The granulation tissue samples were obtained from two groups of patients during the course of routine periodontal surgery. Group I consisted of six patients, 12-20 years of age, who presented with a diagnosis of localized periodontosis, and with infrabony pockets deeper than 8 mm. Group II consisted of 12 patients, 20-63 years of age, who presented with a diagnosis of advanced chronic periodontitis, also with infrabony pockets greater than 8 mm (Fig. 1).

The infrabony pockets and their granulation tissue contents were exposed via full thickness buccal and lingual flap reflection (Fig. 2 and 3). The surgery was performed under local anesthesia (block and

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infiltration) using Xylocaine hydrochloride 2% with 1:100,000 epinephrine.

The granulation tissue which was housed within the osseous walls of the infrabony defect was carefully removed with a small sharp curette, washed in three rinses of saline until little or no reddish discoloration was discernible, frozen within 30 minutes and stored at -70C. Prior to aqueous tissue extraction, the tissue was thawed at room temperature, minced finely and transferred to ice-water-cooled glass tissue grinders. With small amounts of cold distilled water being added repeatedly, the specimens were homogenized thoroughly under constant cooling until homogenous whitish fluid was obtained. Aliquots of the homogenate were then lyophylized. The lyophylized tissues were reconstituted and the immunoglobulin concentrations of the tissue extracts were determined using low level radial immunodiffusion plates (LC Partigens, Behring Diagnostics, Somerville, N.J.) and WHO serum standards as reference (as previously described).[19]

RESULTS

Granulation tissue from the six patients in Group I (periodontosis) showed a wide range in concentrations of IgA, IgG, and IgM. As seen in Table 1, values expressed as µg Ig/mg dry tissue ranged from 0.09 to 1.48 for IgA, 1.44 to 14.10 for IgG, and 0.00 to 0.76 for IgM. There was also a wide variability in concentrations for samples from different sites within the same patient. A smaller range in the immunoglobulin levels was shown for the granulation tissue from 17 patients in Group II (advanced periotontitis). The immunoglobulin concentrations (Table 2) ranged from 0.03 to 0.58 for IgA, 1.18 to 5.55 for IgG, and 0.00 to

0.75 for IgM. These values are also indicative of the variability in concentrations observed throughout the study.

Comparison of mean immunoglobulin levels between groups (Table 3) showed a statistically significant increase ($p < 0.05$) for IgG in the granulation tissue from the periodontosis patients. No significant differences in IgA or IgM levels were detected.

DISCUSSION

Until recently, periodontosis was considered a degenerative disorder of the periodontium resulting in rapid destruction of the supporting tissues in young individuals. Clinically, one usually sees only small amounts of supragingival plaque and little sign of gingivitis, thereby promoting the longstanding concept that inflammation is not playing a vital role in this disease.

Waerhaug,[3,4] however, has shown that there may be a severe chronic inflammation in the soft tissue adjacent to plaque and extensive loss of tooth attachment even though there is little or no supragingival plaque and little or no visible gingival inflammation. He found an extremely high degree of correlation between loss of attachment and downgrowth of plaque indicating a strong cause and effect relationship, and concluded that periodontosis is a destructive juvenile periodontitis caused by rapidly advancing nonmineralized subgingival plaque, previously escaping the attention of clinicians.

It is generally accepted that microorganisms are ultimately responsible for provoking alterations characteristic of periodontitis[20,21] and that bacteria initiate immunopathologic inflammatory reactions

which lead to tissue alterations.[22] Several investigators[23-25] have shown that periodontosis pockets were dominated by gram negative anaerobic rods, indicating a significant microbial difference from the flora isolated from periodontitis. Subgingival microbial plaque accumulation invariably results in an acute inflammatory response with infiltration of lymphocytes and plasma cells with a possible outpouring of immunoglobulins.[22,26] The results of our study support the concept of periodontosis as an inflammatory disease with the local amount of immunoglobulins in the granulation tissue from periodontosis patients equal to (IgM, IgA) or greater than (IgG) the immunoglobulin levels in granulation tissue from infrabony pockets of advanced periodontitis.

An interesting observation pertaining to the periodontosis group was that the tissue samples removed from different defects of the same patient revealed different immunoglobulin levels (Table 1). This was particularly evident for IgA and IgG. Such a variability of Ig concentrations has been reported[19] between tissue specimens with the same degree of inflammation from different areas of the mouth in the same patient.

It is tempting to speculate that this variability in Ig levels may be an indication of an intermittent destructive activity within the lesion, and the ensuing exacerbation may be characteristic of the enhanced aggressiveness of the periodontosis lesion, and that the variability observed for the tissues from different areas from the same patient would be indicative of the destructive or inactive status

of a particular lesion at the time of its excision.

Although a statistically significant increase in mean levels of IgM in granulation tissue from periodontosis and periodontitis granulation tissue was not observed, IgM was *detected* in five of six (or 86%) of the periodontosis granulation tissues compared to 63% in periodontitis granulation tissues, and as previously reported[19] only 40% for normal gingiva. Failure to consistently detect IgM in gingival tissues has been reported earlier.[19,27]

It is possible that the increased detection rate of IgM in the periodontosis granulation tissues is representing an enhanced response to an increased number of gram negative bacteria in the area of periodontal destruction. Possible protective effects of IgM against gram negative organisms may, however, be offset by dysfunction of polymorphonuclear leukocytes manifested by a reduction in phagocytosis and the diminished response to chemotactic stimuli as reported by Lavine[16] and Cianciola *et al.*[17]

Evidence has been presented that IgM is a more efficient activator of complement than IgG[28] and it is therefore probably more efficient in killing gram negative organisms. If this is the case, then our evidence would support the concept of a normal production rate of immunoglobulins in patients with periodontosis, but a deficient cellular immune response. The likelihood of a functionally intact specific humoral response is supported by the fact that in all other respects, persons with periodontosis appear normal. In addition, according to a hypothesis by Lehner[12] explaining the immune response in periodontosis,

the activity of B lymphocytes and subsequent production of antibodies remain intact.

As stated earlier, Lehner *et al.*[11] noted significantly increased levels of serum IgM, IgG, and IgA in patients with periodontosis. This appears to parallel our findings of increased immunoglobulin concentrations in the granulation tissues of periodontosis patients when compared with normal tissue immunoglobulin levels reported by Gross *et al.*[19] The concentration of immunoglobulins in the area of tissue destruction would seem to be a better correlate of the contribution of immunoglobulins (protective or injurious) to the periodontosis lesions than Ig levels in the serum.

Although we did not detect a significant increase of mean IgM level in tissue from periodontosis patients, there was an increased detection rate. However, a significantly increased IgG tissue level was found. When the tissue immunoglobulin concentrations of periodontosis patients were compared to values obtained for normal tissue[19] there was an increase in all three immunoglobulin classes with IgG showing the most significant increase. The increases observed were similar to those shown for gingival tissue in adult periotontitis.[19]

It should be emphasized, however, that the statistical analysis of the differences in immunoglobulin concentrations in granulation tissues from periodontosis and periodontitis patients was performed on a rather small number of specimens and therefore the results should be considered as preliminary findings. Difficulties in obtaining samples from infrequent cases of periodontosis did not allow us to include a

greater sample population in our study.

No normal controls were used in this study since by definition granulation tissue does not exist as a normal tissue. For this reason we chose to compare the Ig content of granulation tissue of two distinct periodontal diseases.

It is hoped that additional studies will be conducted by other investigators and that our findings and findings by others may contribute to a better understanding of the etiology of periodontosis.

SUMMARY

In recent years evidence has been appearing in the literature indicating that the immune response may be playing an important role in the initiation and progression of the periodontal diseases. It was felt that research should be continued in this area, and a study was initiated to determine the IgA, IgG, and IgM concentrations in the granulation tissue removed from deep infrabony pockets of patients with periodontosis and advanced periodontitis. The data revealed a considerable variability in concentrations of the immunoglobulins. Comparison of mean immunoglobulin levels between the periodontosis and periodontitis groups revealed a statistically significant increase ($p < 0.05$) for IgG in the granulation tissue from the periodontosis group. The possible significance of these findings are discussed.

* * * *

Commercial materials and equipment are identified in this report to specify the investigative procedures. Such identification does not imply recommendation or endorsement or that the materials and equipment are necessarily the best available for the purpose. Furthermore, the opinions expressed herein are those of the authors and are not to be construed as those of the U. S. Army Medical Department.

Table 1. Immunoglobulin levels in granulation tissue from periodontosis patients (µg/mg dry tissue).

Patient	Age	IgA	IgG	IgM
1	12	0.12	1.81*	0.49
		0.09	1.62*	0.00
		0.12	2.35*	0.00
		0.10	1.44	0.00
		Avg: 0.11	Avg: 1.81	Avg: 0.12
2	16	1.48	3.01	0.00
		0.58	5.85	0.22
		0.12	9.51	0.00
		0.91	11.67	0.00
		0.84	8.26	0.34
		Avg: 0.79	Avg: 7.66	Avg: 0.11
3	16	0.12	0.74	0.00
		0.43	2.87	0.00
		0.87	14.10	0.12
		Avg: 0.47	Avg: 5.90	Avg: 0.04
4	16	0.49	8.03	0.76
5	20	0.16	2.22	0.00
6	18	0.01	2.92	0.00
		0.02	3.73	0.73
		0.13	6.60	0.00
		Avg: 0.05	4.66	0.17
			Avg: 4.48	Avg: 0.23
Mean		0.35	5.02	0.21
Standard Error		±0.12	±1.08	±0.11

*Determined by electroimmunodiffusion

Table 2. Immunoglobulin levels in granulation tissue from periodontitis patients (µg/mg dry tissue).

Patient	Age		IgA		IgG		IgM
1	20		0.20		1.27		0.25
2	34		0.11		2.58		0.03
3	35		0.03		4.48		0.00
4	36		0.23		5.55		0.00
5	43		0.38		1.57		0.21
			0.61		1.42*		0.33
			0.26		1.36		0.20
		Avg:	0.42	Avg:	1.45	Avg:	0.25
6	43		0.03		1.80		1.27
7	43		0.04		5.14		0.00
8	45		0.27		4.40		0.00
9	47		0.33		3.40		0.00
10	50		0.30		4.77		0.75
11	62		0.49		1.33		0.38
12	63		0.26		3.15		0.50
13	47		0.17		2.61		0.32
14	44		0.21		1.66		0.00
			0.15		1.22		0.24
		Avg:	0.18	Avg:	1.44	Avg:	0.12
15	49		0.18		1.18		0.00
16	58		0.58		4.18		0.66
17	40		0.20		1.39		0.11
Mean			0.24		2.95		0.27
Standard Error			±0.04		±0.37		±0.09

*Determined by electroimmunodiffusion

Table 3. Mean concentration of immunoglobulins in Periodontosis and Periodontitis granulation tissue (µg/mg dry tissue).

	IgA	IgG	IgM
Group I Periodontosis	0.35 ±0.12	5.02* ±1.08	0.21 ±0.11
Group II Periodontitis	0.24 ±0.04	2.95 ±0.37	0.27 ±0.09

*Significant increase ($p < 0.05$)

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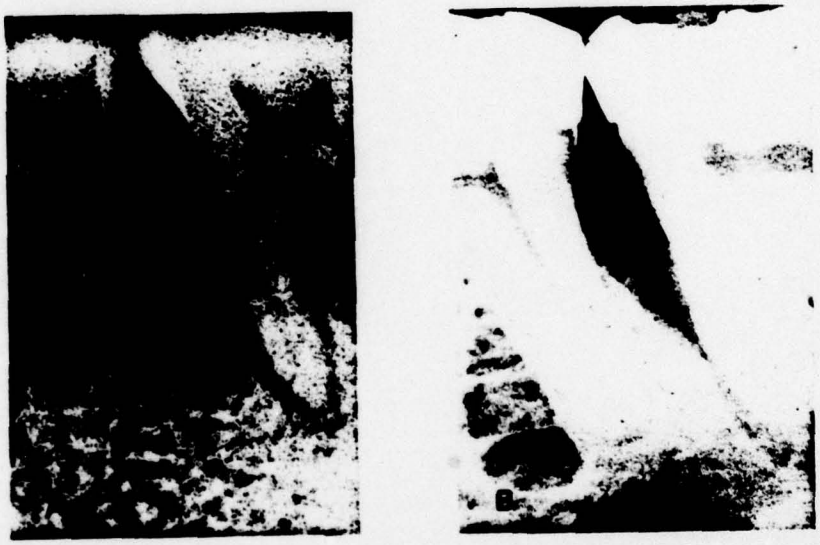

Fig. 1 - Radiographic picture of typical infrabony pockets from which granulation tissue was removed for this study. A. Periodontosis Lesion (note severe "blow-out" type osseous destruction). B. Periodontitis Lesion.

Fig. 2 - Buccal view, following flap reflection, of typical granulation tissue donor site used in this study. The small arrows indicate the buccal osseous rim of the defect; the large arrow points out the granulation tissue which was removed from within the osseous walls.

Fig. 3 - Lingual view of Fig. 2. The small arrows indicate the lingual osseous rim of the defect.